XANTHAN

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Xanthan gum is an acidic, high molecular mass (2–62 MDa) bacterial extracellular polysaccharide (EPS). This EPS is produced as a loosely held extracellular slime by members of the bacterial genus *Xanthomonas*, which consists of plant-associated bacteria. Xanthan gum was first isolated from *X. campestris* pv. *campestris*, the causal agent of black rot of cabbage. The EPS is composed of a branched pentasaccharide repeating unit consisting of D-glucosyl, D-mannosyl, and D-glucuronic acid residues in a molar ratio of 2:2:1, substituted with variable amounts of O-acetyl and pyruvyl residues (Fig. 1) (1,2). The backbone is cellulosic because it consists of β-1,4-linked glucose. The unique rheologic properties and stability of xanthan gum has led to its use as a thickener, emulsifier, and stabilizer in food and nonfood industries. In solution, the EPS exhibits pseudoplasticity; the viscocity decreases as the shear rate increases and this decrease is instantaneous and reversible. Xanthan gum is insensitive to a broad range of temperature, pH, and electrolyte concentrations and is highly viscous at low concentrations. Xanthan gum was first commercialized in 1964 and approved for use in foods by the U.S. Food and Drug Administration in 1969. It is commonly found in a variety of food products, including creamy salad dressings, sauces, and juices. Currently it is estimated that 10,000 to 20,000 tons of xanthan gum are commercially produced worldwide (1,3).

The molecular genetics of xanthan gum biosynthesis has been extensively researched (1,4). These studies have centered on *X. campestris* pv. *campestris* strain NRRL B-1459, the strain used for commercial production of xanthan gum. The genes required for biosynthesis of the necessary sugar nucleotides (UDP-glucose, GDP-mannose, and UDP-glucuronic acid) are located in a 35.3-kb gene cluster. The *xanA* gene encodes an enzyme with phosphoglucomutase and phosphomannomutase activities. The *xanB* gene encodes for a bifunctional enzyme with phosphomannose isomerase and GDP-mannose pyrophosphorylase activities. A homologous gene, *algA*, occurs in *Pseudomonas* species, where it encodes for a bifunctional enzyme with identical activities necessary for the production of the EPS alginate (5). The 35.3-kb region also includes the genes that encode for UDP-glucose pyrophosphorylase and UDP-glucose dehydrogenase. The genes required for synthesis of the lipid-bound repeating unit, for polymerization and export have been located in a continuous 16-kb gene cluster called *gum* that is not linked to the *xan* locus. The *gum* region contains 12 open reading frames (*gumB* to *gumM*) and is expressed as a single operon. The pentasaccharide repeating unit is built up sequentially on a polyprenol phosphate carrier in the cytoplasmic membrane using the appropriate sugar nucleotides and five specific glycosyltransferases.

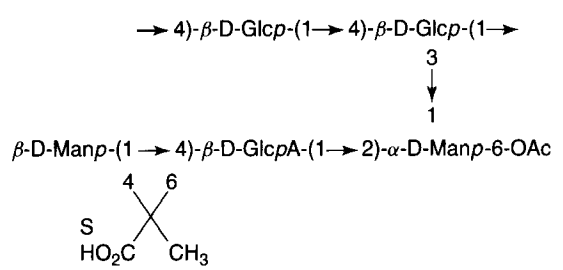

Figure 1. Structure of the repeating unit of xanthan gum. Glc, glucose; Man, mannose; GlcA, glucuronic acid (all three sugars are present in the pyranose form); OAc, O-acetyl group.

The regulation of xanthan gum synthesis is complex, but has not been studied in depth. Several positive and negative regulatory genes have been identified as well as two component regulatory systems (6–8). Xanthan gum production is further regulated by a catabolite gene activator protein, a global regulator of gene expression similar to the catabolite gene activator protein of *E. coli* (9,10).

Production of xanthan gum *in planta* is thought to play a role in the interaction of xanthomonads with plants. The presence of xanthan gum in infected leaves of host plants has been demonstrated both by scanning electron microscopy employing xanthan-specific monoclonal antibodies and by isolation from infected host tissues followed by confirmation of chemical identity (11,12). Several studies with pleiotrophic mutants have indicated that nonmucoid variants of xanthomonads are of reduced virulence, whereas variants that overproduce xanthan gum are of increased virulence. More recently several stable *gum* mutants of *X. campestris* pv. *campestris* were demonstrated to be of reduced virulence (13). Xanthan gum production in leaves is thought to be primarily responsible for the water-soaked appearance of lesions induced by several xanthomonads on their respective hosts. Production of xanthan gum may also be important for symptom production by xylem-inhabiting xanthomonads that cause vascular wilts. Other functions proposed for xanthan gum include, acting as an adhesin for bacterial attachment to wound sites on leaves, aiding in the invasion of leaves via hydathodes, and masking the presence of the invading bacterium in the host leading to a lessened or delayed host defense response (14–16).

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